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ABSTRACT

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Neurocommunicology:

A Model and Implications for Communication Fear Interventions[1]

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Abstract

Current models of communication fear and avoidance are analyzed (i.e., genetic, social and interactional). A new model is proposed, linking language, imagery, emotion and behavior to complex neurological systems in the brain. Evidence for the model is taken from several related fields of study, especially neurolinguistics, neuropsychology, neurophysiology and neuropharmacology. Implications for the understanding, measurement and efficacy of treatment interventions designed to reduce communication fear are advanced.

Key Words: Neurocommunicology, communication apprehension, neuroscience, treatment.

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Neurocommunicology: A Model and Implications for Communication Fear Interventions

The January 2000 edition of *Communication Education* provided a forum for an age-old debate in the social sciences, pitting nature against nurture. Researchers who study communication fear and related constructs, such as communication apprehension, reticence, social anxiety and shyness, have argued over which global category, nature or nurture, explains more of the variation in individual differences. Although scholars agree that both nature and nurture influence emotional predisposition to some degree, the current debate focuses on which of the two is the prevalent cause (Beatty & McCroskey, 2000a, 2000b; Condit 2000a, 2000b; McCroskey & Beatty 2000).

Researchers adopting the nature paradigm assert that genetic inheritance explains the majority of the variation in individual differences associated with communication fear. Researchers adopting the nurture paradigm, in contrast, propose that anxious feelings associated with communication are primarily learned responses emerging from differing social environments. Others have attempted to reduce the dialectical tension between nature and nurture, asserting an interactional model in which both genes and social learning explain individual differences in communication fear. Because interactional models embrace both nature and nurture, the question as to which of the two causes prevails is not as important.

At first glance, it is difficult to find limitations with an interactional model—what other causes, besides genes and environment, could be responsible for individual differences in communication fear? A careful look at the question itself, however, reveals the limitation. Asking what the causes or *inputs* are that determine the differences in communication fear overlooks the fact that our neurological processes serve *simultaneously* as *inputs*, *processes* and *outputs*.

The purpose of this paper is to offer an alternative conceptualization of communication fear, shifting attention away from *input-output* models and focusing instead on an *input-process-output* model that places neurological activity at the crossroads of genotype, environment, language, imagery, emotion and behavior. Advancing a neurological model serves to generate new and intriguing questions regarding the experience of communication fear. Condit (2000a) recommended that communication scholars "continue to develop paradigms and methods that operate at and integrate multiple levels of analysis from the individual to the social to the biological" (p. 23). To that end, a neurological model of communication fear provides an integration of the individual, social and biological domains, functioning as a catalyst for future research questions.

The manuscript is separated into three sections: (1) critique of genetic, social learning and interactional models of communication fear, (2) explanation of the neurological model, and (3) implications regarding the understanding and treatment of communication fear.

Critique of Genetic, Social Learning and Interactional Models

There are three broad categories of models used to explain individual differences in communication fear: genetic, social learning, and interactional models. Genetic models examine how biological inheritance (i.e., genotype) shapes emotional processes involved during interaction. Social learning models focus on the mechanisms by which individuals come to associate communication with anxiety, such as negative thoughts, conditioned anxiety, and skill deficit. Interactional models offer a fusion of genetic and social explanations, conceptualizing communication fear as both inherited and learned.

Genetic Models

The genetic model suggests an input-output model of communication fear, in which genetic variation causes individual differences in communication fear (Beatty, McCroskey and Heisel, 1998). According to the model, genetic variation causes structural and neurochemical differences across individuals, which, in turn, cause individual differences in communication fear. Evidence cited to warrant the claim of inheritance emerges from three bodies of literature: (1) genetic polymorphism, (2) animal studies, and (3) behavior genetic research (i.e., twin comparisons).

Genetic polymorphism. The majority of genetic research on anxiety has focused on the impact of different forms of the same gene, known as genetic polymorphism. Lewin (1985) defined polymorphism as "the simultaneous occupance in the population of genomes showing allelic variations (as seen either in alleles producing different phenotypes)" (p. 690).

Research, to date, suggests that individual differences in anxiety are explained, in part, by a genetic polymorphism regarding the length of a neurotransporter for serotonin, which allows for communication between the neurons that comprise the serotonin systems in the brain. Although this line of research shows promise, models have minimal explanatory power (7 to 9% of variation explained). Keaten, Sakamoto, and Pribyl (2000, p. 22) explained:

> Many questions arise concerning the discussion of genetics and personality. So far, research has suggested that even though an "anxiety gene" has been found, the explanatory power of that single gene is still rather weak. One explanation for the low explanatory power is because of both the sheer number of genes involved in the trait and the limited resources to find, map, and determine their influence on personality.

In sum, studies of genetic polymorphism have offered a precise although weak explanation regarding genetic inheritance and anxiety.

Animal studies. Neuroscientists have attempted to explain the biological mechanisms of fear by comparing the neurological processes of the human brain to those of other animals. Rat and monkey brains are most often studied because of their anatomical, organizational and neurochemical similarities to humans (Ratey, 2001, p. 22).

Jeffrey Gray (1991), for example, developed a theory of temperament based upon neurophysiological characteristics of rats. He described and catalogued the specific neurological structures and functions involved in emotional behavior (Gray, 1982a, 1982b, 1991; Gray, Feldon, Rawlins, Hemsley, & Smith, 1991; Gray, Owen, Davis, & Tsaltas, 1983). Gray (1991) proposed that emotional behavior is the product of three dimensions, corresponding to specific neurological systems. The first system, the behavioral inhibition system (BIS), is associated with anxiety, "a state in which one responds to threat (stimuli associated with punishment or nonreward) or uncertainty (novelty) with the reaction, 'stop, look, and listen, and get ready for action'" (Gray, 1991, p. 110). The behavioral approach system (BAS), the second system, is thought to be associated with reward seeking behavior. The BAS is activated by conditioned stimuli associated with reward (or the termination or omission of punishment), resulting in a release of dopamine from neurons in the ventral tegmental area (Gray, 1991; Gray et al., 1991). The final system, labeled the fight-flight system (F/FLS), is activated by unconditioned aversive stimuli (defensive aggression or escape behavior). The reaction of the fight-flight system determines an individual's level of defensiveness, manifest emotionally as anger.

Laboratory experimentation on animal emotions provides a level of precision and control that is not possible with human subjects. Researchers, for example, study neural systems by removing an area of an animal's brain or severing select neural projections then they measure the effect of the procedure on the animal's behavior. Obviously, these experimental practices are not used on human subjects.

The critical limitation of animal studies of emotion, however, rests in the neurological incompatibility between animals and humans. Human brains are different morphologically and functionally from brains of other animals (Calvin, 1996; Deacon, 1997; Pinker, 1994). Unlike rats, cats, and monkeys, humans use a system of agreement that links sounds and shapes to physical objects, motion, and images. Furthermore, humans create complex linguistic worlds that categorize and contextualize emotion. Although the limbic systems of humans are very similar to other mammals (i.e. hypothalamus, hippocampus, and amygdala), language provides a unique and vital survival advantage, endowing humans with the ability to *act* rather than just *react* (LeDoux, 1996, p. 175)

Although other animals may possess sophisticated systems of communication (i.e., dolphins, honey bees and chimps), humans are unique in their ability to create, negotiate and propagate symbols (Deacon, 1997). Some theorists carry the argument further, asserting

that symbolic activity is a human *instinct* (Pinker, 1997) and that we are born with a universal grammar (Chomsky, 1972, 1975, 1980). Regardless of whether or not symbolic behavior is innate, humans communicate in a unique way that allows us to categorize and attach meaning to physiological states.

Humans also create, manage and interact in linguistic worlds. Social context, a unique human construction, allows for the symbolic manipulation of a physical environment through the creation and negotiation of meaning. Public speaking, for example, is a social context replete with imagery, language, emotion, rules and stories. When individuals use language to understand a physiological response to a communicative behavior (i.e., labeling the sympathetic response of the autonomic nervous system experienced while speaking to an audience as "stage fright"), we activate stored images and actions associated with the context, which, in turn, explain both the appropriateness and meaning of our response.

In summary, the experience of fear in animals is not compatible with the experience of fear in humans. Animal models omit the vital role of language in human neurological processes. The vertical systems of the human brain connect language (left lateral lobe), emotion (limbic systems) and behavior (motor cortex), providing physical evidence that the neurological activity involved in human emotion is unique (Luu & Tucker, 1998), making the analogy between rats and humans problematic.

Behavior genetic studies. Separating genetic inheritance from environmental factors requires specialized research designs. The most frequently used design involves comparing monozygotic twins (MZ; one fertilized egg) to dizygotic twins (DZ; two fertilized eggs) who are raised together (Strelau, 1998, p. 243). Monozygotic twins have an identical genotype, while dizygotic twins share approximately 50% of their genes according to the polygenetic theory. Adoption designs compare the similarities between an adopted child, the child's biological parents, and the adoptive parents. Adoption-twin designs compare MZ and DZ twins raised together as well as MZ and DZ twins raised apart. Although the adoption-twin research design is regarded as the most powerful a very small number of twins are reared apart (Loehlin, 1992).

Jan Strelau, for example, proposed that "temperament" is the result of biological evolution, comprised of those behavioral patterns that are the product of the nervous system. His studies of twins suggest that "emotional reactivity" is the partial product of heredity (Strelau, 1995).

Twin comparisons provide a natural yet controlled method for isolating the effects of both genes and environment on human behavior. Twin comparison studies are limited, however, unless three nontrivial assumptions can be met:

> First, it is assumed that gene-environment correlation and interaction are negligible for the trait in question. Second, it is assumed that the parents of the twins are not correlated for the trait...Third, it is assumed that the effects of nonadditive genetic factors such as genetic dominance or epistasis are negligible (Loehlin, 1992, p. 11).

Condit (2000a) pointed out a significant problem with the first assumption (i.e., no correlation between genes and environment) in adoption twin designs, which biases heritability estimates:

> Twins "reared together" may have an environment that is more similar in some respects than twins "reared apart" but the familiar dynamics of "within family" variation may actually be greater than between family variation in effect. (p. 15)

Correlations between identical twins also may be inflated by design because researchers operationally define personality using self-report questionnaires (Condit, 2000a, p. 11).

Twin studies are limited further because they do not look at genes alone. Identical twins share, for the most part, the same physical characteristics or phenotype. Studying MZ twins, consequently, means studying two individuals with *nearly identical* physical attributes, such as body type, facial symmetry, height, and hair color. DZ twins, in contrast, are not "nearly identical" in their physical attributes because they inherit different genotypes. Because physical attributes affect numerous social phenomena, such as persuasiveness, self-esteem and antisocial behavior (see Knapp & Hall, 1997, pp. 197-248), estimates of genetic inheritance may be inflated as a result of the interaction between phenotype and social environment.

Social Learning Models

Social learning models propose that modeling and conditioning explain individual differences in communication fear. These models assume each individual is a blank canvas at birth awaiting the brush strokes of her or his social environment. According to social learning models, communication fear is caused by cognition (negative thoughts), affect (conditioned anxiety), behavior (skills deficit) or some combination of the three (see Ayres & Hopf, 1993; Glaser, 1980).

Cognitive models operate from the premise that people suffer from anxiety because of the way in which they think (Ellis, 1962; Fremouw, 1984; Meichenbaum, 1977). People experience anxiety because they repeat irrational thoughts (Ellis, 1962; Ellis & Harper, 1975), lack the cognitive ability to cope with irrational thoughts (Meichenbaum, 1977), or because they maintain negative mental images associated with communication (Ayres, Hopf, & Ayres, 1997).

Affective models are based upon the assumption that people have learned to associate the process of communication with anxiety (Friedrich, Goss, Cunconan, & Lane, 1997; McCroskey, 1997; McCroskey & Beatty, 1986; Richmond & McCroskey, 1992). They have become conditioned, through experience and/or observation to believe "that speaking can lead to embarrassment or other forms of psychological discomfort" (Friedrich, Goss, Cunconan, & Lane, 1997, p. 317).

Behavioral models propose that anxiety is the product of a skill deficit (Glaser, 1981; Keaten & Kelly, 2000; Kelly, 1997; Kelly, Phillips, & Keaten, 1995; Phillips, 1968, 1984, 1986, 1991, 1997). Because one does not know how to communicate effectively or competently (Phillips, 1991), a person anticipates negative outcomes when communicating, which triggers an anxious response (Kelly, Phillips, & Keaten, 1995).

Researchers adopting the social learning model have discovered pervasive patterns of thought, emotion and behavior associated with communication problems (for a review, see Daly, Caughlin, & Stafford, 1997, pp. 21-71). Despite the consistent results, the question still remains as to whether the thoughts, conditioning or behavior cause communication fear or whether they are the byproducts of biological predisposition. McCroskey and Beatty (2000) asserted that the enduring and highly stable character of negative emotions toward communicating swings the pendulum toward biological predisposition:

> [R]easearch indicates that, while a few people can change a great deal, most people can't change much. Furthermore, much of the change which we can observe is due to unfolding genetic programming, not individual volition. (p. 3)

McCroskey and Beatty (2000) imply that an inertia regarding negative feelings toward communication stems from genetic predisposition.

The likelihood of social learning as the sole cause of communication fear is low because of an extensive body of experimental studies that demonstrate the efficacy of pharmacological intervention when treating emotional disorders such as social anxiety, depression and schizophrenia. Neuropharmacological studies provide compelling evidence as to the importance of the biochemical processes involved with thought, emotion and behavior (Benson, 1994).

Interactional Models

Interactional models assert that both genes and environment serve as causes of communication fear. Cloninger, Przybeck, and Svrakic (1991), for example, proposed that personality is the result of the interaction between genotype and environment, which is in stark contrast to theorists such as Gray (1982a) and Eysenck and Eysenck (1985) who viewed personality (temperament) as largely the result of genetic inheritance. Cloninger et al (1991) explained the unique relationship between genetic structure, attitudes, opinions, beliefs, and society:

> Genetically regulated neurophysiological processes determine basic personality dimensions which direct tendencies to activate, maintain, or inhibit behavior and influence the acquisition of attitudes, opinions, and beliefs from the range in a given society.

In essence, Cloninger and colleagues argued that attitudes, opinions and beliefs stem from the interaction between genetic structure and culture (society). Culture or society constrains personality because the individual is exposed to

a *finite* set of behaviors, bounded by a restricted set of attitudes, opinions, and beliefs.

Interactional models provide a rich synthesis of genetic and social explanations. Communication fear, for example, can be conceptualized as the product of a dynamic interaction between biological predisposition and pervasive patterns in one's social environment. The limitation of interactional models, however, emerges from an *input-output* structure where genetic predisposition and social learning cause differences in feelings toward communicating. Specifying inputs ignores the dynamic and complex neurological *processes* that make human communication possible.

In brief, interactional models look for the genetic and social inputs that cause communication fear, overlooking the dynamic and complex neurological processes that serve simultaneously as inputs, processes, and outputs. Furthermore, interactional models do not incorporate the anatomical, functional and neurochemical processes that generate, modify and limit human communication.

The next section offers a model of communication that focuses on neurological processes, located at the intersection of genotype, emotion, language, imagery, and environment. The intent of the model is to offer a conceptualization of communication that is grounded in the biochemical systems and structural architecture that allows for interaction in a dynamic symbolic environment. Moreover, the model serves as a catalyst for "questions about the interactions of human biology and culture in the complex process of human communication" (Condit, 2000b, p. 35).

Neurocommunicology

Neurocommunicology refers to the study of how complex neurological processes produce, shape and limit communication behavior in a social, linguistic and physical environment. The preponderance of evidence upon which the model is built comes from several innovative technologies used to map neurological activity: (1) computerized tomography (CT scan) a technique where several narrow beam x-rays are taken of the brain, recording differences in cell densities, (2) positron emission tomography (PET) where subjects ingest a glucose based radioisotope that allows instruments to measure blood concentrations in the brain, and, therefore, neural activity, (3) functional magnetic resonance imaging (fMRI) which imposes a magnetic field onto the brain to detect atomic density and changes in oxygenation, and (4) magneto encephalogram (MEG) measures the magnetic fields produced by the electrical currents of neurons. These four techniques (i.e., CT, PET, fMRI and MEG) are noninvasive technologies that provide detailed spatio-temporal maps of neural activity.

The model is also built upon neuropharmacological research, specifically the biochemical processes by which neurons communicate. Neurotransmitters (i.e., acetylcholine, serotonin, norepinephrine and dopamine) are one method by which neurons communicate, serving to link synapses that comprise functional systems of the brain. For example, acetylcholine ($C_7H_{17}NO_3$), the first discovered neurotransmitter, connects neurons thought to be involved in the process of learning and associative memory. Serotonin ($C_{10}H_{12}N_2O$), implicated in the systems that regulate mood, has been the subject of numerous psychiatric studies. Norepinephrine ($C_8H_{11}NO_3$), the neurotransmitter thought to be responsible for the "fight or flight response," affects both mood and emotion. Dopamine ($C_8H_{11}NO_2$) regulates movement and, when overabundant in the limbic system, is linked to schizophrenia. Research has shown a consistent relationship between levels of neurotransmitters and personality characteristics such as novelty seeking, harm avoidance and reward dependence (Cloninger et al., 1991).

Although these four transmitters (e.g., acetylcholine, serotonin, norepinephrine and dopamine) represent a small minority of neurochemicals, they are the subject of numerous studies because of their involvement in learning, emotion and behavior. Although neurotransmitters play a role in linking functional neural systems, models that advance a one-to-one correspondence between a neurotransmitter and a behavioral predisposition (i.e., serotonin imbalance causes depression) may be oversimplifying the complex and dynamic processes that comprise neural activity.

The model of neurocommunicology places the vertical yet integrated structure of the

[Insert Figure 1 about here]

human brain at the center of the communication process (see Figure 1). The model is built upon

six key links, each vital to our understanding of communicative behavior.

Link One: Genotype and Brain Development

Lewin (1985) defined genotype as "the genetic constitution of an organism" (p. 686). Rather than equating genotype to a "blueprint" for the construction of an individual, genotype refers to the *potential* for the development of an individual that is determined by a person's genetic constitution. The genotype consists of hereditary information that may be passed onto children even though the parent may not acquire his or her genetic potential. For example, an individual may possess the genetic potential to be six feet tall. Whether or not the individual achieves his or her genetic potential, however, is a function of the environment.

The structure of the human brain, encoded in genotype, reflects a long history of evolution (Ratey, 2001). In fact, brain development in the fetus recapitulates the stages of human evolution. During initial development, we form a "reptilian" brain (subcortical region or brainstem), which controls functional systems such as sleep, respiration, body temperature and automatic movement. Built upon the reptilian brain is the mammalian brain or limbic system, which enhances movement, creates memory and produces emotion. The final layer, the cortex, is built upon the limbic system. The cortex refines the lower functions, promotes integration and facilitates language. The three levels (i.e., cortical, limbic, and subcortical) constitute the vertical phylogenetic organization of the brain. Although they are distinct in location and function, these three regions are linked inextricably by complex synaptic and neurochemical systems (Luu & Tucker, 1998; Pert, 1997).

Genotype does not, however, determine the outcome of development because the embryo is surrounded from the moment of conception by a unique biological environment. The characteristics of the environment, especially in the first six weeks after conception, affect the ability of fetal cells to carry out their instructions for development. Ratey (2001) explained the profound interaction between instructions (genotype) and environment during the later stages of fetal development:

> There is a drop from about 200 billion neurons to 100 billion. This widespread cell death is normal, for it eliminates the wrong and weak connections that could inhibit efficient and proper brain function. This is a classic example of the incredible resourcefulness of evolution, which makes us highly adaptable creatures. It also points to the fact that even at the very beginning of development the brain is a social organ: where there is no connection, there is no life. (p. 26)

Neural pathways, either built by design or structured by environment influence, are reinforced through nutrition. Therefore, a newborn's brain already contains complex neural networks that reflect the dynamic interaction between genotype and environment.

After birth, the brain continues to grow, creating new synapses and eliminating others through atrophy. Critical periods of development arise "when the connections for a function are extremely receptive to input. Once the window closes, neural connections are pruned down to the most efficient, according to how much they are used" (Ratey, 2001, p. 40). Critical periods, for example, have been identified for the acquisition of phonemes and grammatical structures (Ratey, 2001).

Link Two: Genotype and Language

Human DNA contains instructions for building key areas involved in language. Other primates, such as monkeys, "lack this left lateral language area: their vocalizations...utilize a more primitive cortical speech area above the corpus callosum" (Calvin, 1996, p.79). Some theorists, most notably Noam Chomsky, argued further that our DNA contains instructions for developing a *universal grammar* (Chomsky, 1972, 1975, 1980). Similarly, Pinker (1994) asserted that we are born with a language instinct, making language different from other human abilities:

> Language is a complex, specialized skill, which develops in the child spontaneously, without conscious effort or formal instruction, is deployed without awareness of its underlying logic..." (p. 18).

Although the possibility of a universal grammar or a language instinct is intriguing, research data are inconclusive. Deacon (1997), for

instance, refuted the idea of a language instinct suggesting instead that humans are biased toward *learning* language:

> Rather than a language organ or some instinctual grammatical knowledge, what sets human beings apart is an innate bias for learning in a way that minimizes the cognitive interference that other species encounter when attempted to discover the logic behind symbolic reference...(p. 141).

Lieberman (2000) proposed that although the neural circuitry necessary for language is innate, language acquisition (i.e., phonemes, words, and grammar) is a learned process. He rejects the existence of a language module, proposing, as an alternative, a functional language system (FLS) that integrates the vertical structures of the brain:

> The correct model for the functional organization of the human brain is not that offered by "modular theorists...a set of petty bureaucrats each of which controls a behavior and won't have anything to do with one another." The neural bases of human language are intertwined with other aspects of cognition, motor control and emotion. (p. 2).

Data taken from CT, PET, fMRI and MEG scans suggest that language is not a localized brain function. Rather it is the product of complex neural networks that incorporate cortical, limbic and subcortical systems (Stemmer & Whitaker, 1999).

Link Three: Imagery, Language and Neurological Activity

Although genotype directs the composition of the brain, physical energy in our environment also creates and alters synaptic structure, especially when processed repeatedly. When we encode fragments of sensory information into neural networks, we develop sets of coherent and identifiable images. Although our neural networks of images allow us to perceive our environment as a whole, information gathered by the sensory organs is fragmented (Ratey, 2001). When we stand in front of an audience, for example, our neural networks regarding the event serve to create a coherent picture puzzle of the audience, room, etc. despite the fact that many pieces of the sensory puzzle are missing.

The richness of language as a source of information stems from neural networks that span both the vertical and lateral structures of the brain (for a review of tomographic research on language, such as CT, PET, fMRI and MEG scans, see Demonet, 1998; Dronkers & Ludy, 1998; Fields & Troster, 1998; Luu & Tucker, 1998; Papanicolaou, Simos, & Basile, 1998; Segalowitz & Chevalier, 1998a, 1998b; and Whitaker, 1998). Language systems are structured upon several important social agreements, such as segmentation and categorization of physical and social phenomena (semantics), guidelines concerning how to feel about these categories (prosody and frequently associated words), rules for organizing categories (syntax) and discourse telling us how and why things occur (Damasio & Damasio, 1999). The implicit social agreement reflected in language structures allows for vital human activities, such as the coordination of action, sharing of resources and development of identity and meaning (Baars, 1997; Deacon, 1997; Lieberman, 2000; Pearce & Cronen, 1980; Pinker, 1994; Shimanoff, 1980).

Recent research in neuroscience suggests that the architecture of the human brain promotes symbolic expressions. Demasio and Demasio (1999) explained three networks that produce linguistic devices, such as metaphor:

> First, a large collection of neural systems in both the right and left cerebral hemispheres represents nonlanguage interactions between the body and its environment, as mediated by varied sensory and motor systems...it also creates another level of representation for the results of its classification...Second, a smaller number of neural systems, generally located in the left cerebral hemisphere, represent phonemes, phoneme combinations, and syntactic rules for combining words...A third set of structures, also located largely in the left hemisphere, mediates between the two. (pp. 30-31)

Because neural networks connect images and actions with speech production, "symbolic representations such as metaphor can easily emerge from this architecture." (Damasio & Damasio, 1999, p. 34). In essence, sensorimotor images are married synaptically to

linguistic constructions, generating linguistic devices, especially metaphor.

The combination of imagery and language allows for the creation of social context (see Baars, 1997, pp. 115-129). When we ground new information in a context, we activate language systems that guide perception, emotion and behavior. Language networks, therefore, serve to construct an ordered world of social contexts. For example, when we activate language networks that are connected to "fear", serving to categorize the sympathetic response of the autonomic nervous system, we also evoke all the accoutrements of language—vocal inflections hinting at limbic arousal (prosody), associated concepts (harm, threat, etc.), syntax telling us the object of the fear, and discourse that explains how and why fear occurs (e.g., "butterflies in my stomach"). LeDoux (1996) contended that language makes the human experience of emotion unique: "feelings will be different in a brain that can classify the world linguistically and categorize experience in words than in a brain that cannot" (p. 302).

Language systems are also adaptable, evolving continually to meet the demands of a structured yet unpredictable physical and social environment (Sankoff, 1980). Although our brains are shaped by language (Stemmer & Whitaker, 1999), our brains also shape language to satisfy physical, psychological, social and existential needs (Epstein, 1990). Once we learn how to interact with the world linguistically (i.e., semantics, syntax, discourse), we seek incoming information that conforms to those known patterns. When current categorizations are not found useful new language systems emerge, which, in turn, categorize future sensory information (Deacon, 1997).

Link Four: Brain and Behavior

Human behavior is the product of cortical, limbic and subcortical systems. At the most basic level, all behaviors are muscle movements produced by motor neurons. The basal ganglia and the cerebellum refine muscle movements by adjusting force, timing and execution. Integration with sensory systems in the cortex allows muscle movement to be skilled, adaptive and functional (Kolb & Wishaw, 1996). Because motor neurons connect, either directly or indirectly, to other functions, behavior can be viewed as the juxtaposition of muscle movement, physical energy in the environment, stored images, language systems, and limbic activation.

Face to face communication behavior reflects the multifunctional and vertical structure of brain systems. If we say, "Nice weather we are having", we produce a symphony of intricate muscle movements (controlling respiration, vocal tract movement, tongue and mouth movement), produce vocal inflections (pitch, rate, volume and quality), exhibit facial expressions that indicate limbic arousal, and employ language, revealing the structures that compartmentalize, categorize, and explain our social environment. Because these systems operate simultaneously, our behavior can be viewed as the manifestation of sensory, emotional and linguistic systems. To study behavior, language, imagery and emotion separately, therefore, is to ignore the nature of the neurological systems that generate these phenomena.

Link Five: Sensory Organs and the Brain

Perception, in neurological terms, can be defined as the "subjective experience of the physical energy in one's environment" (Kolb & Wishaw, 1996, p. 118). Perception is not, however, the passive absorption of environmental stimuli (i.e., sensation). Rather, perception is an active process that is rooted in preconceived notions of context:

> The clearest proof that perception is more than sensation is the transformation of the same sensory stimulation into totally different perceptions and the fact that perceptions are affected by the context of the sensory input. (Kolb & Wishaw, 1996, p. 119)

Functional systems in the brain determine the information to which the sensory organs attend. Our sensory organs also serve a vital limiting function, sheltering us from the tidal wave of surging environmental stimuli (Kolb & Wishaw, 1996).

Information attended to by the sensory organs is projected to working memory, located primarily in the principle sulcus, which allows for "short-term activation and storage of symbolic information" (Goldman-Rakic, 1999, p. 92). A projection to long-term memory (i.e., principle sulcus to hippocampus) allows information stored in short-term memory to be compared to learned associations. Working memory is also

part of an elaborate network that connects, "major sensory, limbic and premotor areas of the cerebral cortex" (Goldman-Rakic, 1999, p. 97). A region of long-term memory (hippocampus), for example, projects to the center of emotional memory (lateral nucleus of amygdala), infusing emotional information associated with the context. In essence, information attended to by the sensory organs is analyzed and potentially stored by comparing the new information to learned associations and emotional memory, resulting frequently in the activation of the motor systems.

Link Six: Genotype and Phenotype

Phenotype is defined as "the appearance or other characteristics of an organism, resulting from the interaction of its genetic constitution with the environment" (Lewin, 1985, p. 689). Phenotype refers to the observable or physical characteristics of an individual, such as height, eye color, and hair color. Recall that genotype refers to the inherited potential of an individual whereas phenotype refers to the actual physical state of the individual, which is a product of both genotype and environment. Lewin (1985) offered an explanation of the essential difference between phenotype and genotype: "Visible or otherwise measurable properties are called the phenotype, while the genetic factors responsible for creating the phenotype are called the genotype" (p. 25).

A person's height can illustrate the difference between genotype and phenotype. If a person possesses the potential to be six feet tall (genotype) but if the availability of nutritious food (environmental characteristic) was scarce during the growth phase of the individual, the result might be an individual who grows to only five feet tall (phenotype). While separated for ease of explanation both the genotype and environment work in concert to develop the organism (Lewin, 1985)

Phenotype is similar, but not identical, to the construct of appearance. Appearance includes the subjective and culturally guided process of perception, whereas phenotype refers to *objective* biological characteristics. The metaphor of appearance as a "second skin" exemplifies the elemental difference between phenotype and appearance. Phenotype is a first skin (observable physical characteristics), whereas perception of phenotype (appearance) is a second skin, which is largely the result of social covenants regarding the meaning of physical attributes (Burgoon, Buller & Woodall, 1996, pp. 48-60).

Model Summary

The most complex human activity, communication, is located at the bustling crossroads of genotype, phenotype, sensory images, language, behavior and environment. At conception, we inherit instructions for a vertical and phylogenetically structured brain. As biological processes unfold in a physical and social environment, a phenotype emerges. Part of our initial development involves the formation of neural systems necessary for language acquisition, consisting of countless connections between cortical, limbic and subcortical regions. Language serves as a multifaceted social instrument, which functions to contain, categorize, contextualize and explain our dynamic environment. Finally, the complexity of human communication behavior echoes the intricacy of the neurological processes that produce it. In summary, communicative behavior is produced, shaped and limited by functional, integrated and adaptive neural systems.

Implications for Communication Fear

Adopting a neurological model of communication has many important implications for both understanding, measuring and coping with communication "fear." Below, some major implications are discussed and research directions are provided.

Uniqueness of Human Fear

Human fear is unique because our language systems modify the experience of emotion through categorization and discourse related to emotion (see Links 2 & 3). The fear of communication, therefore, is different neurologically than fear experienced by other mammals. Humans, unlike other primates, possess the ability to assign symbols to emotions. LeDoux (1996) discussed the exceptional nature of human emotion, focusing on our unique ability to plan emotion. "Emotional plans are a wonderful addition to emotional automaticity. They allow us to be emotional *actors*, rather than just *reactors*" (p. 177). Our unique ability to plan emotion may be due in part to an enlarged prefrontal cortex (LeDoux, 1996).

Human fear is unique, furthermore, because of neural networks that connect nonlinguistic information (i.e., sensorimotor imagery) to linguistic information (i.e., semantics, prosody, syntax, and discursive structures). Because of the synaptic web of emotional, connotative, discursive, and syntactical information conveyed during linguistic interaction, individuals will develop neural structures that predispose them to certain words, images, emotions and behaviors. For example, if a parent talks to a child using the semantic categorization of "public speaking", while simultaneously exhibiting vocal signals of elevated arousal (prosody) and facial expressions symptomatic of fear, the child will build pathways that associate "public speaking" with elevated levels of limbic arousal.

Associating behavioral manifestations of limbic activation (i.e., prosody & facial expression) with semantic referents, a process referred to here as *linguistic conditioning*, occurs despite the fact that the child has *no knowledge* of what a speech entails and no prior knowledge of the semantic categorization "public speech." Linguistic conditioning is viable because temporally associated information creates new pathways, resulting in a neural record of language and its related sensorimotor images. As the linguistic coincidence is repeated, neural pathways are activated and reinforced by parallel pathways, increasing the strength and ease of communication between neurons that compromise the new network.

The implication of linguistic conditioning when helping people with communication problems is to examine language usage in those individuals who experience communication fear, especially metaphor, prosody, syntax and discourse. For example, changing *conceptual metaphors* (i.e., sensorimotor imagery coupled with subjective experience; see Lakoff & Johnson, 1999, p. 45) may help to restructure perception, which may refocus attention and, thus, the interpretation of novel information. Future research might examine the relationship between conceptual metaphors, social context perceptions and the experience of communication fear.

Multifaceted Intervention

Communication researchers often refer to the areas of cognition, emotion, and behavior as if they are distinct and discrete domains. Neurological data (CT, PET, fMRI & MEG scans), however, show that images, language, emotion and behavior are synaptically intertwined and, therefore, inseparable systems (see Link 4). When a concept, such as "fear" is activated its neurological correlates are also activated, such as sensorimotor images, semantic correlates, and emotional memories.

The interconnectedness of neural systems responsible for imagery, language, emotion and behavior, offers a parsimonious explanation regarding the consistent correlations found between cognitive, affective and behavioral measures of communication fear (Daly, Caughlin, and Stafford, 1997; Kelly, 1982). Neurological intertwining of systems may also explain why combination treatments (i.e. cognitive, affective and behavioral) are the most effective for treating public speaking fear (see Allen, Hunter & Donohue, 1989).

Because imagery, language, emotion and behavior are mutually dependent processes they are best addressed as a unified system rather than as separate components. A careful examination shows that the vast majority of intervention strategies integrate these dimensions despite their claims to be grounded in a single domain (e.g., cognition, affect or behavior). Systematic desensitization, for example, teaches muscle relaxation but relies on language to create images of behaviors (i.e., "walking up before the audience"; see Friedrich, Goss, Cunconan, & Lane, 1997, p. 310). Visualization (Ayres, Hopf, & Ayres, 1997) employs a narrative script rather than a sequence of visual images. Visualization scripts (Ayres & Hopf, 1993, p. 33) also include allusions to positive feelings such as "full of confidence" and "feeling very good." Rhetoritherapy (Kelly, Phillips, & Keaten, 1995) uses cognitive structures to modify behavior, asking questions such as what is a *reasonable* goal. Furthermore, goal analysis, a foundational component of rhetoritherapy, requires individuals to assess "positive" and "negative" responses to their communicative behavior (Kelly, Phillips, & Keaten, 1995, p. 270). In short, communication interventions appear to embrace the interconnected nature of functional brain systems despite their labeling to the contrary (i.e., cognitive, affective, behavioral).

Measurement of Communication Fear

A neurological model of communication fear has implications for measurement because communication context is a social construction rather than a physical reality (see Link 3). Communication contexts, such as dyads, groups, and public speeches are linguistic constructions that classify ambiguous sets of events. Public speaking, for example, is a not a monolithic social event. It is, rather, a semantic construction that serves to cluster a nebulous confluence of images, language, emotion and behavior. If "public speaking" were a single event, features such as audience size, audience familiarity, topic familiarity, and seating configuration would not vary. Evidence suggests the contrary. In specific, images, language, emotion and behavior associated with public speaking are subject to great variation (see Daly, Caughlin, and Stafford, 1997). Ayres and Heuett (1997, 1999), for example, have shown that mental images associated with public speaking are highly variable across individuals.

Because of the ambiguous nature of a communication context, instruments designed to measure negative emotion associated with communication (i.e., apprehension, anxiety, fear, reticence) may be more useful if we specify contextual features. We might offer information, for instance, that reflects incorporated neurological systems, such as imagery, semantic categorization, discourse (narrative), emotion and behavior. The following item might serve as an example for measuring the fear of public speaking within a distinct context:

> Imagine yourself as a student in a basic public speaking course with 25 other students. Your first class assignment is to give a three-minute speech on a topic of your choosing. After receiving the assignment, you selected a topic that interests you and you have conducted the appropriate research. Your teacher appears energetic and takes time to speak to each student in the class.
>
> Today is the day you are scheduled to speak. You are scheduled to speak first to your class. You get up to address the class. How do you feel?
>
> Nervous 7 6 5 4 3 2 1 Not Nervous
> Relaxed 7 6 5 4 3 2 1 Not Relaxed
> Tense 7 6 5 4 3 2 1 Not Tense

The above example is one possible way that a self-report questionnaire might embrace the interconnected structure of neural systems (visual images, discourse, behavior and emotion). By demystifying context, we may be able to reduce the inherent ambiguity of linguistic constructions such as "group discussion" and "public speaking." Without contextual information, we may foster a false sense of precision in our measurement, when, in fact, we are gathering information on amorphous linguistic constructions that defy consistent interpretation.

Timing and Intervention

Neurological development, due to both genotype and environment, is a temporal phenomenon (see Link 2 and Link 5). As neurological processes are repeated over time, activated neural networks are nourished and neighboring neurons are recruited to strengthen the network (Kandel & Hawkins, 1999). Because repetition over time strengthens neural networks, individuals who have developed and employed dysfunctional systems (language, imagery, emotion, and behavior) over a long period of time will require more time to untangle and disable dysfunctional networks to form new and productive neural systems. Interventions, such as visualization, systematic desensitization, cognitive restructuring, rhetoritherapy, etc., might honor this neurological limitation. In specific, we may need to acknowledge and act upon the negative relationship between the continued use of dysfunctional neural networks and treatment efficiency. By assessing the depth of dysfunctional networks (language, imagery, emotion and behavior) we may obtain more accurate estimates regarding the time and energy needed for effective communication intervention. Finally, practioners in our field may consider the efficacy of interventions at early stages of communication development, such as the preschool and elementary levels. Although housing communication intervention programs in higher education institutions may be practical, it may also limit severely our ability to help individuals overcome deeply rooted communication difficulties.

Limbic Arousal and Treatment

Research shows a clear curvilinear relationship between associative memory and limbic arousal (LeDoux, 1996). In particular, moderate levels of arousal (i.e., moderate levels

of norepinephrine) tend to enhance associative learning and the retrieval of stored information:

> The levels of NE [norepinephrine] in the brain and body are related to how well an animal or human learns or remembers. To take a simple example, people tend to remember events that were associated with strong emotions such as anger, fear or grief. These emotional states typically involve increased blood levels of NE released from the adrenal gland and probably in the NE brain circuits as well. (Thompson, 1993, p. 141)

The suggestion that a moderate state of limbic arousal produces optimal learning has important implications for helping students who suffer from communication problems.

Current interventions differ as to the preferred level of limbic arousal during treatment. Some interventions, for example, promote low levels of limbic arousal (relaxation; systematic desensitization; Friedrich, Goss, Cunconan, & Lane, 1997, p. 309) whereas other interventions promote moderate to high levels limbic arousal ("full of energy, full of confidence;" visualization; Ayres & Hopf, 1993, p. 33). Other interventions do not specify a preferred state of limbic arousal, such as rhetoritherapy (Kelly et al., 1995), cognitive-orientation modification (Motley, 1997) and skills training (Kelly, 1997). The relationship between limbic arousal and treatment efficacy is relatively unknown and, therefore, provides a fertile ground for future research.

Metaphor and Treatment Intervention

Metaphor is a powerful linguistic device that embraces our neurological architecture (see Link 3). Metaphor serves as the bridge between sensorimotor information and subjective experience (Lakoff and Johnson, 1999). Because of their compelling nature, metaphors might help people to understand and cope with their communication problems. Ideally, metaphors would compel productive behavior through the use of memorable images that promote constructive behaviors. Furthermore, the degree to which the information offered in our interventions is compelling will predict, perhaps, the extent to which individuals repeat what they have learned.

If we wanted to develop a metaphor, for example, that emphasizes the importance of mental repetition when restructuring negative thoughts, we might envision the brain as a dense jungle (Ratey, 2001). We also can build upon the jungle metaphor by equating thinking with walking through the jungle. By linking complex neurological processes such as thinking with stored sensory images (jungle) and motor behavior (walking) we might form a memorable and potentially beneficial association. Here is a sample script that might be offered during a cognitive restructuring intervention:

> Think of your brain as a vast jungle. In this thick and lush jungle there are many paths leading in countless directions. Some paths have been traveled frequently (highway) and others are traveled infrequently (trails). There are also paths in this lush jungle that have not been trod. Paths, whether highways or trails, are analogous to certain thought patterns in your brain. When you repeat a thought you construct a road, regardless of whether that road leads to good places (productive thought) or bad places (irrational or negative thought).
>
> If we depart from the highway (frequent thought) and blaze a new path (novel thought) through the thick jungle, we face resistance and uncertainty, which can make us feel uncomfortable and often anxious. When we complete our first trip through an uncharted section of the jungle, however, we leave a path that makes traveling the same way a little easier each time (the more often you repeat a thought the easier it becomes to think it). If we never go back to the new path (fail to repeat a new thought), the jungle consumes the path quickly and it is lost. If we walk the path regularly, however, we tame parts of the jungle and future trips will require less exertion, permitting us to move more quickly. Frequent travel turns our path into a road (new thoughts become part of our neurological structure). Our old, negative thoughts become unused roads that the jungle eventually consumes, making future travel very difficult (negative thoughts diminish).

If the metaphor is compelling (brain=jungle & thinking=walking), students will remember the necessity of walking repeatedly on the new path (i.e., repeating visual images, positive thoughts, or coping statements). In short, creating metaphors that represent complex neural processes accurately might help students to understand their communication problems and promote repetition of treatment, both of which may improve treatment outcomes.

Summary

Neurocommunicology is defined as the study of how complex neurological processes produce, shape and limit communication behavior in a social, linguistic and physical environment. Communication behavior, according to the model, is the product of the dynamic interaction between genotype, imagery, language, emotion, phenotype, and behavior. Advanced imaging technology (i.e., CT, PET, fMRI, & MEG) supports this integrated perspective.

Adopting a neurological model has many implications and provokes a host of new questions regarding the conceptualization, measurement, and treatment of communication fear. The human experience of fear is unique because of neural pathways that connect eclectic sources of information, such as language, imagery, emotion and behavior. According to the model, linguistic behavior plays a central role in the understanding of negative emotion associated with communication.

In closing, Francis Bacon developed a metaphor of the human brain (i.e., mind) as an "enchanted glass" that transforms our physical and social environment:

> For the mind...is far from the nature of a clear and equal glass wherein the beams of things should reflect according to their true incidence; nay, it is rather like an enchanted glass.

Reframing communication as a complex neurological phenomenon may be a critical first step in helping us to understand the enchanted glass that transforms experience, emotion and communicative behavior.

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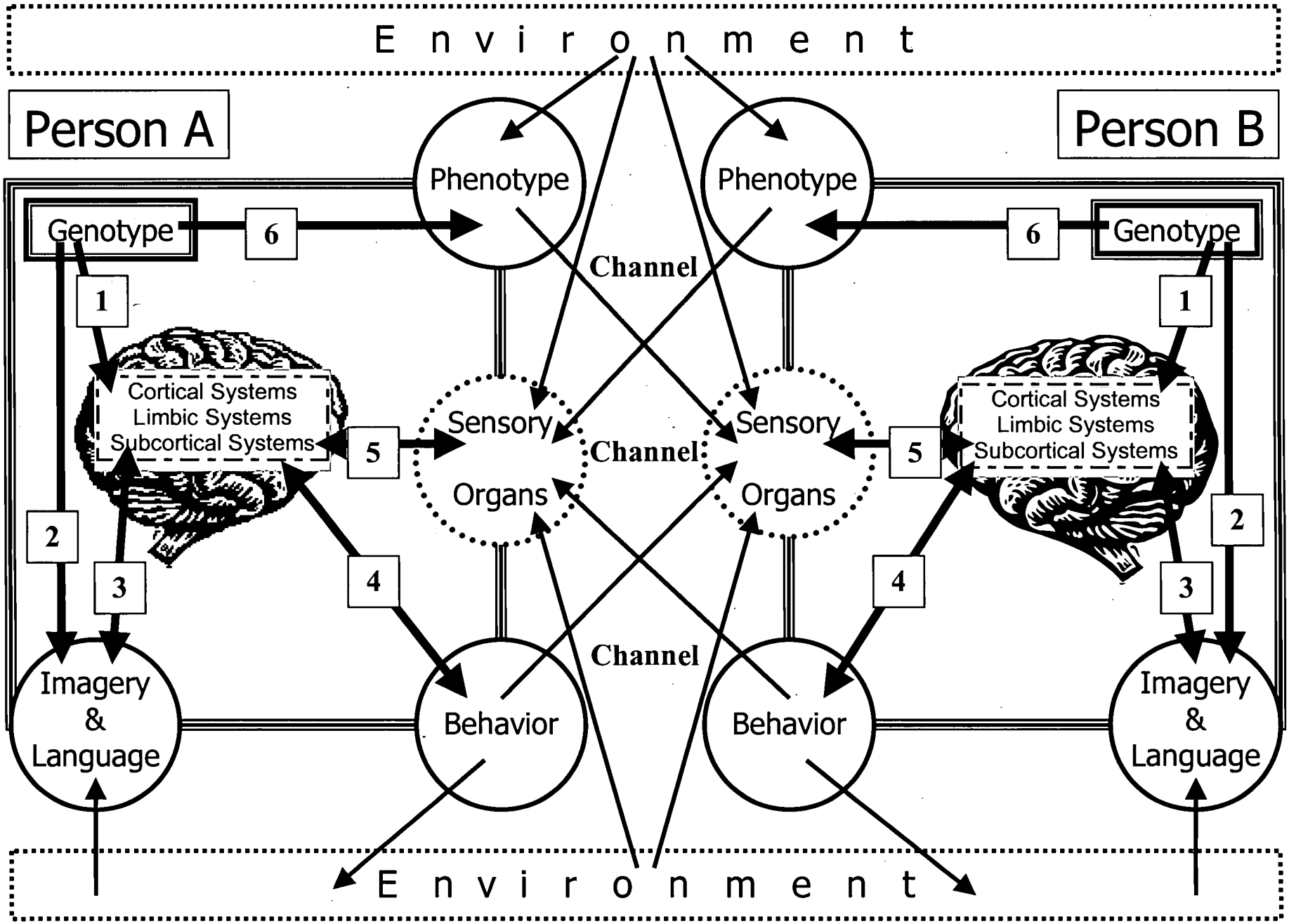
Figure 1

Neurocommunicology



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